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FATIGUE MODELS FOR APPLIED RESEARCH IN WARFIGHTING

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RUNNING HEAD: Warfighter Fatigue Model

Abstract

The United States Department of Defense (DOD) has long pursued applied research concerning fatigue in sustained and continuous military operations. In 1996, Hursh at Science Applications International Corporation (SAIC), under an Army sponsored contract, developed a simple homeostatic fatigue model and, working with Precision Control and Design, programmed the model into an actigraph to give a continuous indication of performance. Based on this initial work, the Army conducted a study of 1-week's restricted sleep in 66 subjects with multiple measures of performance, termed the Sleep Dose-Response Study (SDR). This study provided numerical estimation of parameters for the Walter Reed Army Institute of Research (WRAIR) Sleep Performance Model (SPM), and elucidated the relationships among several sleep-related performance measures (6). Concurrently, Hursh at SAIC extended the original actigraph modeling structure and software expressions for use in other practical applications under sponsorship of the Natick Research and Development Center (31) and later with the Air Force Research Laboratory (AFRL). The model became known as the Sleep, Activity, Fatigue, and Task Effectiveness (SAFTE) Model, and Hursh has applied it in the construction of a Fatigue Avoidance Scheduling Tool (FAST) under an AF SBIR awarded to NTI, Inc. (16). This software is designed to help optimize the operational management of aviation ground and flight crews, but is not limited to that application. This paper describes the working fatigue model as it is being developed by the DOD laboratories, using the conceptual framework, vernacular, and notation of the SAFTE model (16). At specific points where the WRAIR SPM may differ from SAFTE, this is discussed. Extensions of the SAFTE model to incorporate dynamic phase

adjustment for both transmeridian relocation and shift work are described. The unexpected persistence of performance effects following chronic sleep restriction found in the SDR study necessitated some revisions of the SAFTE model that are also described. The paper concludes with a discussion of several important modeling issues that remain to be addressed.

INDEX TERMS: Sleep, Fatigue, Circadian Rhythm, Performance, Model, Cognitive Throughput, Sleep Inertia, Sleep Deprivation

FATIGUE MODELS FOR APPLIED RESEARCH IN WARFIGHTING

Introduction

The United States Department of Defense (DOD) has long pursued applied research concerning fatigue in sustained and continuous military operations. Lead DOD laboratories are the Walter Reed Army Institute of Research (WRAIR) in Silver Spring, Maryland, the Naval Health Research Center (NHRC) in San Diego, California, the Air Force Research Laboratory (AFRL) at Brooks AFB, Texas, and the U. S. Army Aviation Research Laboratory (USAARL) at Fort Rucker, Alabama. Research teams at these locations are responsible for investigating fatigue-related impairment of cognitive readiness," for developing countermeasures to fatigue, and for providing guidance to the Services in the management of fatigue.

A three-process, quantitative model was initially conceived in the 1980s, jointly by WRAIR and Scientific Applications International Corp. (SAIC), in an attempt to estimate a relationship between crewmen's sleep and the delivery of artillery rounds on target (30). During the 1990s, WRAIR focused on the study of sleep per se as a determinant of cognitive performance, which contributed to refinements of the original model from data obtained from studies of total and partial sleep deprivation (7). WRAIR sponsored the development of an actigraph with an embedded sleep model, and Hursh at SAIC developed a simple homeostatic fatigue model and, working with Precision Control and Design, programmed the model into an actigraph to give a continuous indication of performance. These efforts suggested the need for a large-scale study of partial sleep deprivation to fill a major knowledge gap between normal sleep

and total sleep deprivation. A study was undertaken of 1 week's restricted sleep in 66 subjects with multiple measures of performance, termed the Sleep Dose-Response Study (SDR). This study provided numerical estimation of parameters for the WRAIR Sleep Performance Model (SPM), and elucidated the relationships among several sleep-related performance measures (6). Concurrently, Hursh at SAIC extended the original actigraph modeling structure and software expressions for use in other practical applications. Work sponsored by the Natick Research and Development Center focused attention on the development of the fatigue model for incorporation into the Integrated Unit Simulation System (IUSS), a simulation of solder performance under hypothetical combat scenarios (31). With support from the AFRL's Warfighter Fatigue Countermeasures (WFC) Program, Hursh further developed the Sleep, Activity, Fatigue, and Task Effectiveness (SAFTE) Model, and has applied it in the construction of a Fatigue Avoidance Scheduling Tool (FAST) under an AF SBIR awarded to NTI, Inc. (16). This software is designed to help optimize the operational management of aviation ground and flight crews, but is not limited to that application. Current laboratory collaborations between NHRC and AFRL, additional field data collection by both groups, and studies of sleep deprivation and pharmaceutical effects in the Army laboratories all promise to add quantitative input and validation for model development. This combined progress led to a meeting among DOD principal investigators in January 2002, for the purpose of more closely coordinating their research and converging parallel efforts in modeling development. Given their common origin, the WRAIR SPM and AFRL/SAIC SAFTE models do not differ greatly. Structural differences are minor. The key distinction between the two approaches is the temporal perspective of their application. The WRAIR SPM model, with its roots in an actigraph-based monitoring technology, attempts to take *prior,* measured sleep history of individuals to estimate *current*

cognitive capacity, or "readiness," of both the individual and the crew or group in which he/she operates. It may be used to provide feedback to the individual who may need sleep, to allow selection among candidate individuals or units for a particular operation, or to provide a weighting function for performance in higher order models of operational scenarios. On the other hand, SAFTE is applied to hypothetical or *prospective* work/sleep schedules in order to identify potential performance problems, and to optimize operational planning and management. Clearly these perspectives are complementary and overlap considerably and can share a common model of sleep and performance prediction. In addition, the SAFTE model was elaborated with a fourth process that modulates the sleep reservoir capacity during chronic sleep restriction to account for findings from recent chronic sleep restriction studies showing slower than expected rebound of performance following recovery sleep. The SAFTE model has also been enhanced to account for circadian shifts due to transmeridian crossings versus shift work changes.

This paper describes the working model as it is being developed by the DOD laboratories, using the conceptual framework, vernacular, and notation of the SAFTE model (16). At specific points where the WRAIR SPM may differ from SAFTE, this is discussed. This model is intended to be a tool for the operational components of the Services; that is, its framework is heuristic, and the research focus is toward application. In the background are a number of basic research efforts, supported by government, industrial, and academic enterprise, which will be cited by the authors but not discussed in the depth they deserve. Nonetheless, such efforts both add to the body of knowledge upon which a valid, practical tool can be constructed, and impose important theoretical and practical constraints. In order to plan future studies leading to useful and accurate predictions, in DOD laboratories and elsewhere, basic issues must

be considered, and are discussed critically in the context of the present model. Hopefully, this paper will help guide our laboratories in coordinating their research, and allow the reader to assess the status of our applied research as it progresses toward a transition to practical applications.

The conceptual architecture of the SAFTE model is shown in Figure 1. The core of this model is schematized as a Sleep Reservoir, which represents sleep-dependent processes that govern the capacity to perform cognitive work. Under fully rested, optimal conditions, a person has a finite, maximal capacity to perform, annotated as the Reservoir Capacity, Rc. While one is awake, the actual "contents" of this reservoir are depleted, and while asleep, they are replenished. Replenishment (Sleep Accumulation) is determined by Sleep Intensity, and Sleep Quality. Sleep Intensity is in turn governed by both the Time-of-Day (Circadian Process) and the current level of the reservoir (Sleep Debt). Sleep Quality is modeled as its continuity, or conversely, Fragmentation, in part determined by external, real-world demands, or requirements to perform. Performance Effectiveness is the output of the modeled system. The level of Effectiveness is simultaneously modulated by Time-of-Day (Circadian) effects and the level of the Sleep Reservoir. Transient post-sleep decay of performance is modeled by the term Inertia.

[Figure 1 here]

The foregoing terminology has been selected to provide operational users of the model an intuitive grasp of the processes involved. SAFTE is a three-process, quantitative model similar to that suggested by Folkard and Åkerstedt (18), Achermann and Borbely (1), Åkerstedt and Folkard (2), and Jewett and Kronauer (33). The modulation of reservoir volume essentially represents the homeostatic regulation of wakefulness, involving two subprocesses with respect to

performance capacity (equivalent to their S process). The second process is the major influence of circadian rhythms (process C). The third process involves "sleep inertia" (process W). The following discussion will take up the individual components of the model in some detail.

Process 1: The Homeostatic Process

Wakefulness – Reservoir Depletion

The Performance Use function is a mathematical formula describing the rate at which cognitive performance capacity declines during continuous wakefulness. SAFTE expresses this function in terms of an equation for a straight line, equation 1:

$$P = K \cdot t, \quad (1)$$

where P is Performance Use, or reservoir depletion over a period of time, t. The model pegs the Reservoir Capacity, Rc, at 2880 arbitrary units, and the default value for K, the slope of this line, is 0.5 unit per minute. Thus, after 4 days (5760 minutes) of continuous sleep loss, the reservoir will be fully depleted.

The rationale for both linearity and the value for the decay slope (about 1% per hour awake) derives from a straight-line fit of cognitive throughput data obtained during 72 hours of total sleep deprivation. In that study, performance declined by approximately 25% for every 24 hours of total sleep deprivation (46). The residual data from this treatment show a clear circadian rhythm (Figure 2). The SAFTE model with a linear Performance Use function

combined with a two-frequency circadian process (see below) can fit the data of Figure 2 with an R^2 of 0.89.

[Figure 2 here]

The Performance Use function is a linear approximation of what may be a more complex pattern of decay over time. There remain a number of unresolved issues concerning both its slope and its shape. For instance, data from the SDR study (66 subjects sleeping either 9, 7, 5, or 3 hours per day for 7 days), yielded a straight-line slope of about 0.5% per hour, using a simple reaction time task (PVT) (14) instead of an arithmetic task (6). Whether this twofold difference from the previous estimate is task-specific, or due to other factors (e.g., demographic) remains unclear. Furthermore, other modeling efforts have postulated curvilinear decay functions based on other data sets. Folkard and Åkerstedt (18) use an exponential expression for decreasing alertness (as opposed to performance). A linear approximation of their function over 24 hours yields a slope of about 1.8% per hour. For both alertness and performance, Jewett (32; 33) fits data to a sigmoidal function, reflecting both a delay in the onset of decay after awakening, and a slowing of decay rates after about 36 hours of sleep deprivation. Jewett also suggests that the decay waveform may be influenced by both circadian phase at wake time and by prior sleep debt. Most researchers would probably agree with us that the variance of grouped data tends to increase with the duration, measured in days, of sleep deprivation experiments, which makes precise description of the waveform all the more difficult. For the time being, then, we continue to utilize the linear approximation.

Sleep Accumulation – Reservoir Replenishment

The sleep/restoration function is a mathematical formula describing the rate at which restoration of cognitive performance capacity accrues during sleep. For SAFTE, additions to the reservoir (S) resulting from sleep over an interval of time, t, depend on the Sleep Intensity (SI, or rate of recuperation due to sleep) over that interval, shown in equation 2:

$$S = SI \cdot t. \quad (2)$$

SI (units/minute) varies during the interval such that it is the weighted sum of 1) Sleep Propensity (SP), a function of time-of-day, and 2) the current reservoir deficit, or Sleep Debt (SD), in the reservoir ($Rc-R_t$) as it changes during the interval, multiplied by a feedback factor, *f*. This latter quantity is sometimes referred to as the sleep-wake cycle because it depends on the pattern of sleep and wakefulness. Thus, SI is given by the following sum, equation 3:

$$SI = SP + SD, \text{ where } SD = f \cdot (Rc-R_t). \quad (3)$$

SP incorporates a circadian process, c, and an amplitude factor, a_s (default = 0.55 unit; see below), and *f* has a default value of 0.0026564 min^{-1}. Rc is the reservoir capacity and R_t is the reservoir level at time t. SAFTE incorporates a maximum level of Sleep Intensity, set to 4.4 units/minute. This limit permits an equilibrium state to be reached with as little as 3 hours sleep per day, but not with less. Note, however, that with only 3 hours sleep per day, performance is severely degraded until recovery sleep is obtained.

The proposition that Sleep Intensity is increased by Sleep Debt is a feature recognized by all the models recently offered. For the WRAIR SPM and the homeostatic model of Folkard and Åkerstedt (18), this is explicitly stated as an exponential "recharging" function. The rationale for this derives from observations that the rate at which recuperation occurs during sleep varies continually as a function of extant Sleep Debt. Recuperation at the beginning of the sleep period, when Sleep Debt is relatively high, occurs at a faster rate than at the end of the sleep period, when Sleep Debt is relatively low (38, 28). Whether this is due to shifts of sleep architecture toward more restorative slow-wave sleep in the early hours has been discussed recently by Wesensten, Balkin, and Belenky (48). If expressed as a discrete function, as above, or exponentially, the results of the SPM and SAFTE converge for small intervals, ignoring the circadian process in the SAFTE model. The value of *f* in SAFTE is the reciprocal of the Time Constant of Recuperation in the exponential equation of the SPM, and is equivalent to about 375 minutes (based on a performance throughput measure). This value is derived from earlier studies in which 84 hours of sleep deprivation were interrupted by daily ½ hour naps (7). After the SDR study, the SPM was modified to a much slower rate of recuperation, with a Time Constant of about 1300 minutes (based on a reaction time measure). As with the value for waking decay, this large difference in recovery rate is not entirely understood. It is consistent with the observation that the 3- and 5-hour sleep groups in the SDR did not recover to baseline after three full nights of sleep, a slower rate of recovery than SAFTE would predict suggesting a needed revision, described below.

The circadian component of SI, or Sleep Propensity (SP), essentially postulates that the restorative effect of sleep depends in part on the time-of-day at which the sleep occurs (10, 36). In SAFTE, this is expressed by equation 4:

$$SP = - a_s \cdot c, \quad (4)$$

in units/minute, where a_s is a weighting factor (default = 0.55 unit), and c is the circadian rhythm of body temperature and arousal, which varies between +1 and -1 (see below). For a person taking a normal 8 hours sleep from midnight to 0800, sleep is most intense in the early morning at about 0300. There is a mid-afternoon increase in sleep propensity at about 1600 that coincides with the mid-afternoon dip in alertness and consistent with the observation of increases in sleep related traffic accidents (36). The rhythm of SP is taken to be 180 degrees out of phase with alertness and performance; hence the resulting value is subtracted from the restoration rate due to sleep. Jewett and Kronauer (33) incorporate a similar term in their model that modulates the rate of recovery, arguing that the actual amount of sleep obtained (given equal amounts of time allowed for sleep) varies according to time of day, without implying that changes in sleep architecture (or quality) mediate changes in Sleep Intensity. By the same argument, the SPM omits altogether any correction for circadian effects on sleep quality or quantity, since the SPM is concerned with sleep *as it is actually measured.* This, again, is the key difference between SPM and SAFTE, and SAFTE thus has the advantage of being able to optimize both sleep amount and sleep *timing* for prospective work/rest schedules.

The final influence on Sleep Accumulation results from Sleep Fragmentation. This is expressed as a nonlinear term that has the effect of delaying onset of sleep restoration (by setting SI = 0) at the end of any wake period. This is based on empirical evidence that the early minutes of sleep are generally comprised of Stage 1 sleep (48). By screening out the first several minutes of sleep, the model enhances the effect of fragmented sleep and frequent awakenings, an effect

by which such influences on cognitive performance capacity as age, environmental disruptions, divided work/rest schedules, and sleep pathology are expressed. At present, this delay is set at 5 minutes following each arousal or awakening, based on studies of simulated sleep apnea in which 12 awakenings per hour were equivalent to total sleep deprivation (9). However, it is likely that future research will lead to refinement of this function such that it will be modulated by extant sleep debt and/or time of day. Both SAFTE and SPM contain this factor, and it is closely related to the time-of-sleep discussion by Jewett and Kronauer (33), although not explicitly expressed in their model.

Process 2: Sleep Inertia

Sleep inertia can be described as the delay, after awakening from sleep, before expected levels of alertness and performance resume. The modeling of this transient phenomenon is based on studies of post-sleep performance (13) and of brain metabolism using positron emission tomography (PET) (5). Jewett and Kronauer (33) and Folkard and Åkerstedt (17) both invoke a short-lived exponential deviation from the homeostatic process. The SAFTE model estimates this effect as an exponential discharge function that is invoked for 2 hours after awakening from sleep, whose output is subtracted from the Effectiveness output of the overall model according to equation 5:

$$I = - I_{max} \cdot e^{-(i \bullet ta/SI)}, \text{ for } t_a = 0 \text{ to } 120 \text{ minutes}, \quad (5)$$

where I_{max} is the maximal inertia effect on awakening, set to 5%, and i is the inertia time constant, set to 0.04. Since the time constant is also related to the sleep intensity at time of awakening, SI, sleep inertia will last longer for awakenings that occur during deep sleep, such as early in the sleep period or during sleep periods of individuals carrying a large sleep debt.

Process 3: The Circadian Process

Performance while awake and the drive to sleep are both controlled, in part, by a circadian process (41, 17). Studies of performance (e.g., reaction time (15)), alertness ratings (42, 22), measures of the tendency to fall asleep (e.g., multiple sleep latency tests (11, 40, 45); see also Lavie (37)), and body temperature (22, 42) indicated that the underlying circadian process is not a simple repeating sine wave. Performance and alertness reach a major peak in the early evening, about 2000, and fall to a minimum at about 0400. There is a secondary minimum in the early afternoon, about 1400, and a secondary morning peak at about 1000. Correlated with this pattern is a varying tendency to fall asleep that reaches a peak at about the same time performance and alertness reach their minima. The existence of both a major and a minor peak in performance and two corresponding minima at other times suggests that at least two oscillations are involved in the circadian process (47).

Both SAFTE and SPM estimate this circadian process with a function that is composed of the sum of two cosine waves, one with a period of 24 hours and one with a period of 12 hours. The two oscillations are out of phase producing an asymmetrical wave form: a gradual rise during the day with a plateau in the afternoon and a rapid decline at night that closely parallels published studies of body temperature (22, 42, 24). The circadian rhythm of performance is not

a simple mirror image of variations in body temperature (20, 21). The asymmetrical circadian rhythm combines with a gradually depleting reservoir process resulting in a bimodal variation in cognitive effectiveness that closely parallels published patterns of performance and alertness, described above. The circadian process is represented by equation 6:

$$c_t = \cos(2\pi (T-p)/24) + \beta\cos(4\pi (T-p-p')/24), \quad (6)$$

where T is the time of day in hours, p is the time of the peak of the 24 hour rhythm, p' is the relative time of the 12 hour peak, and β is the relative amplitude of the 12 hour rhythm. Initially, in the SAFTE model, p is set to 1800 hours (6 PM), and is adjusted in a manner described below. The value for p' is 3 hours, and β is 0.5. Parameters derived from analysis of SDR data are implemented in one version of the SPM. These phase values are somewhat later in the day, with a major peak at about 2300 and an afternoon nadir at 1700. Because the SDR study was not designed optimally for elucidation of circadian rhythms, having only four unequally spaced data points during each day, the consensus of our laboratories favors the values used in SAFTE, which better track the timing and amplitude of known circadian processes. Note also that since c is a compound of two cosine functions, the peak of the resulting waveform does not coincide with the peak of the 24 hour component, p; with p equal to 1800 hours and p' set to 3 hours, the peak of the resulting compound is about 2000 hours.

Modification of the Circadian Process by Activity Patterns

When subjects move to another time zone or alter work pattern so that sleep and work occur at different times of day, the internal circadian oscillator that controls body temperature and alertness shifts to this new schedule. During the period of adjustment, subjects experience performance degradation, disrupted mood and feelings of dysphoria, called circadian desynchronization or "jet lag" (35, 24, 29). The SAFTE Model mimics this process and automatically adjusts the phase of the circadian rhythm to coincide with the activity pattern of the subject. This feature is critical for the accurate prediction of the effects of moving to a new time zone or changing to a new and regular work pattern, such as changing from the day shift to the night shift. The model detects the average time of the awake period and maintains a running average "awake time." The peak of the circadian rhythm has a reliable relationship to the timing of the period of wakefulness. When one moves to a new work schedule or a new time zone, the change in average awake time (relative to a reference time zone) is detected and a new "target phase" is computed. For example, after moving from the central U.S. time zone to Germany, the awake time of the subject advances 6 hours. This causes a gradual shift of 6 hours in the circadian process of the model. In general, a phase advance (eastward time change) takes about 1.5 days per hour of shift (39, 25, 23, 43, 35, 24, 29). The model, therefore, adjusts to the new "target phase" gradually over the course of 9 days. During that time, the performance of the subject will show net degradation due to the desynchronization of the internal circadian process from the new rhythm of work and sleep. Likewise, westerly travel causes a phase delay in the circadian rhythm and takes less time for adjustment, about 1 day per hour of shift (39, 25, 23, 43, 35, 24, 29). Folkard et al. (19) similarly utilize time of awakening as the basis for phase

adjustments, while Jewett and Kronauer (33) emphasize the synchronizing effect of light exposure in their model. It is acknowledged that light exposure may be a fundamental driver for phase adjustment, along with sleep, activity and social cues; however, in practice, light exposure information is normally not available to the planner in advance of an operation. As an approximation, periods of awake activity are normally closely linked to times of exposure to light (either natural or artificial) so that the timing of awake activity coincides with the timing of light exposure and can serve as a reasonable basis for the estimation of phase changes. Limitations of this approximation may occur in situations of continuous low-level artificial light (e.g., aboard submarines or orbiting spacecraft) or when exposure to bright light is deliberately arranged to induce a phase shift (34, 12).

Recently, the SAFTE model has been incorporated into a planning tool called the Fatigue Avoidance Scheduling Tool (FAST) which also includes features to track changes in geographic location and calculated levels of sunlight. In this implementation, the model can detect the difference between transmeridian schedule shifts and shift-work changes at the same location. When a shift-work change is detected, a slower rate of phase adjustment is implemented to reflect the inhibitory effects of both light exposure and social cues. At its extreme, a shift-work induced change in circadian phase may take 2.6 times as long to complete as a comparable transmeridian shift in phase (21).

Combined Processes: Performance Effectiveness

The final output of the SAFTE model consists of a summation of the Homeostatic process (Sleep Reservoir balance) and the Circadian Process (Performance Rhythm), with

transient adjustments for sleep inertia as required. In the WRAIR SPM, these terms are combined differently, by multiplying (modulating) the reservoir balance with the Circadian Process. The SAFTE model is computed as a weighted, additive modulation of the level of performance, expressed as a percent of baseline. Thus, Effectiveness at time t (E_t) is given by equation 7:

$$E_t = 100 \cdot (R_t/Rc) + C_t + I, \quad (7)$$

where I is the transient inertia term; $100 \cdot (R_t/Rc)$ is the Reservoir Level, expressed as % of capacity; C_t is computed from the Circadian Process (c) as follows:

$$C_t = c_t \cdot (a_1 + a_2(Rc - R_t)/ Rc), \text{ where } a_1 = 7\% \text{ and } a_2 = 5\%. \quad (8)$$

The computation of the circadian component (C_t, equation 8) includes a variable amplitude expression that effectively increases circadian modulation of effectiveness with increasing sleep debt (4).

Adaptation to Specific Task Effectiveness

The SAFTE model can predict changes in cognitive capacity as measured by standard laboratory tests of cognitive performance. For example, the model can predict degradation of serial add-subtract throughput during 72 hours of sleep deprivation ($R^2 = 0.89$, data from Thorne, et al. (48)) as well as average cognitive throughput across a series of cognitive tests during 54

hours of sleep deprivation ($R^2 = 0.98$, data from Angus and Heslegrave (3); see below). A modified version of SAFTE (see below) with appropriate parameter settings can predict average cognitive throughput and average psychomotor vigilance (PVT) speed during restricted sleep duration over 7 days ($R^2 = 0.94$, data from Balkin, et al. (6); see below). It is assumed that these cognitive tests measure changes in the fundamental capacity to perform a variety of tasks that rely, more or less, on the cognitive skills of discrimination, reaction time, mental processing, reasoning, and language comprehension and production. However, specific tasks, such as specific military tasks vary in their reliance on these skills, and deficits in cognitive capacity may not produce identical reductions in the capacity to perform all military tasks. It is reasonable to assume, however, that the changes in military task performance would correlate with changes in the underlying cognitive capacity. In other words, if one were to plot changes in military task performance as a function of measured changes in cognitive capacity, there would be a monotonic relationship between the two variables. Therefore, if these two sets of data were available from a test population subjected to sleep deprivation, linear (or nonlinear) regression techniques could be applied to derive a transform function; this transform translates predicted cognitive changes into changes in military task performance. Based on this reasoning, the method for evaluating the effectiveness, discussed previously as the cognitive effectiveness, can be extended to predict variations in any task or component of a task (given appropriate test data) using the generalized Task Effectiveness (TE), equation 9) expression as follows:

$$TE = A\,(R_t/Rc) + B + C1\,[\cos(2\Pi(T-P)/24) + C2(\cos(4\Pi(T-P-p')/24))] + I, \qquad (9)$$

where *A* = linear component slope, *B* = linear component intercept, *C1* = Circadian weighting factor, *C2* = 12 hour weighting factor, and *P* = acrophase of the task. The other factors in the equation (R_t/Rc and I) are as they would be predicted by the SAFTE model for cognitive effectiveness.

Implications of Model Structure

Equilibrium States: If a subject is scheduled to take less than an optimal amount of sleep each night, for example, 4 hours per day, the reservoir initially loses more units during the awake period than are made up during the sleep period. This results in a sleep debt at the end of the sleep period that accumulates over days. However, since the rate of sleep accumulation increases with sleep debt, eventually, the rate of sleep accumulation increases such that 4 hours of sleep equilibrates with the depletion of 20 hours awake. At this point, the reservoir reaches an equilibrium state and no further debt is accumulated, although the initial deficit remains as long as the person remains on this schedule. By the sixth day of the restricted sleep schedule, cognitive performance oscillates about a stable level well below the baseline level achieved with 8 hours of sleep. Minimum effectiveness is about 64% on the seventh day.

Progressive Sleep Debt under Extreme Schedules: The sleep homeostat is not infinitely elastic; there is a limit to the rate of sleep accumulation (sleep intensity), set in SAFTE at 4.4 units per minute. The effect of this is that any schedule that provides less than about 3 hours of sleep per day will not reach an equilibrium state and performance capacity will gradually deplete to zero, although the rate of depletion slows over the first week of restriction as sleep intensity rises to its

maximum level. Under a schedule of only 2 hours of sleep per day, minimum performance declines to about 19% on the seventh day.

Sleep Timing: The SAFTE model is sensitive to the time of day of the sleep period. The performance of an individual given 8 hours of sleep per day starting at 1200 (noon) each day reaches a peak of 100% at the start of each work period (2000) but rapidly declines during the late night and early morning hours to a strong dip at about 0500. Minimum predicted performance under this schedule is predicted to be as low as 66% compared to minimum performance under a normal sleep schedule of 86%. This alteration in pattern results from two factors. First, sleep intensity is initially less for sleep periods starting at noon. This results in a small accumulated debt that is quickly offset by the homeostatic sleep mechanism. The second, more persistent effect is the circadian oscillator of performance that reaches its minimum in the early morning hours. This pattern has important implications for performance under shift schedules that require daytime sleep. It is well documented that most mistakes on the night shift occur during the early morning hours and this is predicted by the model (8, 27, 26, 44).

Retrospective Validation: The predictions of the model for the effects of total sleep deprivation were compared to an independent set of data reported by Angus and Heslegrave (3). Their results were plotted against the predictions of the sleep model and are shown in Figure 3. All parameters within the model were set to the default values with the acrophase (peak of the 24-hour circadian rhythm) and start time as indicated in the legend. The SAFTE Model predictions for the actual data were exceptionally good ($R^2 = 0.98$).

[Figure 3 here]

Prospective Validation: The results of the sleep dose response study provide an opportunity to conduct a prospective validation of the SAFTE model against a range of sleep conditions between total sleep deprivation and normal amounts of sleep. Figure 4 is a summary of the results of that study showing the average performance across all cognitive tasks as a percent of the performance of the group provided 9 hours to sleep. This group was used for normalization to account for the clear learning effect that occurred with some of the tasks. The heavy lines through the points are the original SAFTE model predictions. The model does a reasonably good job of predicting the average performance during the course of the 7 days of sleep restriction but does not predict the slow recovery seen during the 3 days of recovery sleep.

[Figure 4 here]

Virtually all models would have predicted full recovery of performance following 3 days of recovery sleep. The relatively permanent effect of chronic sleep restriction suggests that some aspect of sleep homeostasis undergoes a gradual change that is slow to recover. In an accompanying paper, researchers from the WRAIR propose a method to account for this effect. Within the context of the SAFTE model, a simple gradual down-regulation of the sleep reservoir capacity (Rc) during chronic restriction can account for this change. A single equation modulates Rc during sleep, equation 10:

$$Rc_{(t)} = Rc_{(t-1)} + t \cdot [k_1 \cdot (1-(SD_{(t-1)}/k_2)) + k_3 \cdot (2880- Rc_{(t-1)})], \quad (10)$$

where $SD_{(t-1)}$, is the sleep debt component of sleep intensity at time t-1, $[f \cdot (Rc_{(t-1)} - R_{(t-1)})]$. Current sleep intensity, SI, is unchanged from equation 3 except that $Rc_{(t)}$ is allowed to adjust according to equation 10. As before, SP is the sleep propensity, the circadian component of sleep intensity. Parameter *f* is the amplitude of feedback in the original model and $R_{(t)}$ is the current reservoir balance. The exact value of *f* is adjusted to a slightly higher value (0.00312) when implementing equation 10 to ensure that a person getting 8 hours of sleep per day is in balance. Based on the SDR study, the limit of SI is reduced to 3.4 units per minute. In addition, equation 10 is constrained so that when Rc is restored it may not exceed the full capacity of 2880, as represented in the original version of the model. No changes to Rc occur during awake periods. Good fits to data are achieved with constants about equal to the following:

$k_1 = 0.22$, down-regulation time constant

$k_2 = 0.5$, the reference level for SI regulation (note: normal sleep averages one SI unit per minute of sleep)

$k_3 = 0.0015$, recovery time constant.

Equation 10 functions as follows: the first expression within brackets becomes negative when SD exceeds k_2 and down-regulates Rc according to the rate constant k_1; when SD is less than k_2, then the second expression within brackets tends to gradually restore Rc according to the rate constant k_3. Jointly, this expression tends to down-regulate Rc when sleep intensity is high ($> k_2$) and to restore Rc when sleep intensity is low ($< k_2$). During a normal 8-hour period of sleep, Rc is down-regulated slightly and is restored by the end of the night. During prolonged

periods of restricted sleep, Rc is down-regulated more than it is restored so that a gradual shift in the reservoir "set point" occurs. If we think of SD as a measure of "sleepiness," then this process tends to reduce sleepiness by reducing the difference between the current reservoir level and the reservoir capacity or "set point." During periods of restricted sleep, performance tends to be more severely degraded (compared to the original model) because the reservoir reaches equilibrium at a reduced set point. During recovery sleep, performance recovers more slowly (compared to the original model) because both the level of the reservoir and the reservoir capacity must be restored.

[Figure 5 here]

The heavy lines in Figure 5 are the predictions of the modified SAFTE model optimized for average cognitive throughput and using the parameters listed above for equation 10. This version of SAFTE makes identical predictions for total sleep deprivation, so the results in Figure 3 are unchanged. The R^2 for this fit to the mean cognitive performance observed in the SDR study is 0.94.

[Figure 6 here]

Figure 6 displays the average PVT speed from the same study shown in Figure 5 (7). The lines in the figure indicate the predictions of the revised SAFTE model optimized for average PVT speed ($R^2 = 0.94$). Results are shown for the baseline, seven experimental days (E1-E7), and the three recovery days (R1-R3). Note that compared to average cognitive throughput, PVT speeds tends to be more severely degraded and the parameters of the SAFTE

model reflect this difference in sensitivity of PVT speed compared to general cognitive throughput.

Discussion: Critical Issues

All models of sleep and performance have shortcomings, including the SAFTE model. The importance of those limitations depends on the application. Two major limitations are that the model does not provide an estimate of group variance about the average performance prediction and it does not incorporate any individual difference parameters, such as age, morningness/eveningness, or sleep requirement for full performance. These individual characteristics may be relatively unimportant if the application of the model is for prediction of average group performance or for design of a generic schedule to be used by an entire work force. For these applications, ordinal predictions are sufficient to decide which of several alternative schedules is best or to decide if average performance at some future time is expected to be at an acceptable level. If the purpose is to judge a particular person's fitness for duty against some criterion level of performance or to predict the level of performance of a particular performance some time in the future, then greater fidelity to these individual variables and some representation of the amount of predictive error would be valuable. In theory, some of these features could be added to the model based on the available literature. Other features, such as age effects and individual sleep requirements, would be difficult to incorporate without extensive additional research.

The performance of all models will also depend on the quality of the data used to establish the sleep history prior to the period of prediction. The WRAIR SPM model was designed explicitly to use actigraph records of sleep and wakefulness as the basis for prediction. The SAFTE model as it is implemented in the schedule prediction tool, the Fatigue Avoidance Scheduling Tool (FAST) can take actigraph data as input to the prediction. Nevertheless, the results of the WRAIR SDR study showing slow recovery from restricted sleep, if replicated and confirmed, suggests that even a week of prior data may not be entirely adequate to account for the long-term effects of chronic sleep restriction. Indeed, these data suggest that most laboratory studies of sleep deprivation or sleep restriction may be flawed because few of them consider the possible contamination of the results by chronic sleep deprivation that might have preceded the laboratory measurements, especially in college students who have often served as the subjects in these experiments.

Military applications of sleep and performance models will require the incorporation of algorithms to predict the effects of pharmacological countermeasures, such as stimulants to extend performance or sedatives to enhance sleep. Some preliminary work has been done to model the effects of d-amphetamine and modafinil on performance in the SAFTE model but the incorporation of these algorithms into a user tool is somewhat premature. Not only do stimulants temporarily improve performance in the face of sleep deprivation effects, they also can interfere with the ability to obtain restful sleep during the period of their arousal effects. Any complete model of the effects of stimulants must represent both the beneficial effects on cognitive performance and the temporary detrimental effects on sleep if attempted immediately after the drug administration. Similarly, any model that attempts to represent the beneficial effects of a

sedative on sleep must also represent any detrimental cognitive effects that follow drug administration if performance, instead of sleep, is demanded of the subject.

[Figure 7 here]

Finally, all fatigue models presume some performance metric as the cardinal standard for prediction. Some models are explicitly designed to predict subjective alertness as measured by a rating instrument; others are designed to measure cognitive performance. For those designed to predict performance changes, some, like the WRAIR SPM, are optimized to predict reaction time performance on the psychomotor vigilance task (PVT), while others were designed to predict performance throughput (correct answers per minute) across a battery of cognitive tests. The SAFTE model has two sets of parameters that can be used to predict either PVT speed or average cognitive throughput. Even if the PVT is used as the standard test, some researchers focus on speed and others focus on the occurrence of lapses, i.e., unusually long reaction times that may represent brief microsleeps that increase in frequency with duration of sleep deprivation. Figure 7 shows that based on the SDR data, there is a linear relationship between lapse probability and the *inverse* of cognitive throughput or PVT speed. Hence, to properly test a cognitive throughput model, such as the SAFTE model, when using lapse data, an inverse transform of the prediction is necessary. Without such a transform, one finds an exponential relationship between cognitive throughput and lapse probability, and this nonlinearity, if not adjusted for, would cause an increase in prediction error with increases in amount of sleep deprivation. Unfortunately, an inverse transform was not applied to the cognitive throughput predictions for two of the scenarios at the Seattle *Fatigue and Performance Modeling Workshop*

in which the performance metric was PVT lapses, and this would naturally have inflated estimates of prediction error.

It may not be possible or desirable to adopt a universally accepted standard for performance measurement, but in the absence of a standard, great care must be taken when applying a model to a performance metric distinct from the one used to design the model. Ultimately, all models will be judged by their ability to make useful predictions of the performance of greatest interest to the user, which is most likely not going to be performance on a standard cognitive test, but rather performance of some job. The greatest challenge facing fatigue modeling is how to bridge this gap between laboratory metrics of performance and performance in the natural environment of work and war.

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